Obstructive Ileus Secondary to Acute Mesenteric Ischaemia: Internal Medicine Perspective

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ABSTRACT

Acute mesenteric ischemia (AMI) could be a rare but potentially life-threatening condition due to poor understanding of the clinical presentation of abdominal pain and the differential diagnosis when it is not suspected and partly because of an unacceptable delay in making the diagnosis. A 59 years old man was brought to the hospital with abdominal pain, accompanied by obstructive ileus and sepsis. An urgent CT-scan showed the feature of intestinal infarction and portal vein thrombus. After giving fluid resuscitation and antibiotic injection, he was consulted to the surgery division and had jejunum resection. Since the etiology of the disease was suspected to be acute mesenteric venous thrombosis, he was given intravenous anticoagulants postoperatively and the condition improved. The clinical diagnosis of acute mesenteric ischemia is troublesome, and in most cases, abdominal pain is the main symptom. Ileus and sepsis are two complications that may mask the initial signs and symptoms of AMI. From the internal medicine's point of view, the proper treatment of this disease is early diagnosis, the rebuilding of blood flows with anticoagulants, surgery division discussion, and post-operative supportive care. The underlying cause should be established to determine long-term management essential to anticipate a repeat.

Keywords**:** *acute mesenteric ischemia, ileus, diagnosis, treatment, internal medicine*

ABSTRAK

Iskemia mesenterika akut (AMI) merupakan suatu kondisi langka tetapi berpotensi mengancam nyawa karena pemahaman yang tidak komprehensif terhadap presentasi klinis nyeri perut dan diagnosis banding ketika tidak dicurigai dan sebagian karena keterlambatan dalam membuat diagnosis. Seorang laki-laki 59 tahun dibawa ke rumah sakit dengan sakit perut disertai obstruktif ileus dan sepsis. CT-scan menunjukkan gambaran infark usus dan trombus vena portal. Setelah diberikan resusitasi cairan dan injeksi antibiotik, pasien dikonsultasikan ke bagian bedah dan menjalani reseksi jejunum. Karena etiologi penyakitnya dicurigai sebagai trombosis vena mesenterika akut, pasien diberi antikoagulan intravena pasca operasi dan kondisinya membaik. Diagnosis klinis dari iskemia mesenterika akut sangat menyulitkan bagi klinisi, dan pada kebanyakan kasus, nyeri perut merupakan gejala utama. Ileus dan sepsis adalah dua komplikasi yang dapat menutupi tanda dan gejala awal AMI. Dari sudut pandang penyakit dalam, pengobatan yang tepat untuk penyakit ini adalah diagnosis dini, restorasi kembali aliran darah dengan antikoagulan, konsultasi dengan divisi bedah, dan perawatan suportif pasca operasi. Penyebab yang mendasari harus dipastikan untuk menentukan manajemen jangka panjang untuk mengantisipasi terjadinya rekurensi.

Kata kunci: *iskemia mesenterika akut, ileus, diagnosis, tatalaksana, penyakit dalam*

INTRODUCTION

Mesenteric ischemia (MI) is a rare medical condition, incidence of 0.1% of all patient visits to the hospital, with a high mortality rate ranging from 24% - 94%.[1] The disease can be divided into acute and chronic MI. Acute mesenteric ischemia is a severe abdominal emergency characterized by sudden disruption of intestinal blood flow that usually results in intestinal infarction and necrosis. This condition is caused by four main causes: acute arterial embolism, acute arterial thrombosis, non-occlusive mesenteric ischemia, and mesenteric vein thrombosis.[2]

Acute mesenteric ischemia (AMI) could be a rare but potentially life-threatening condition due to poor understanding of the clinical presentation of abdominal pain and the differential diagnosis when it is not suspected and partly because of an unacceptable delay in making the diagnosis. The clinical manifestation of the end stage of AMI consists of diffuse peritonitis, ileus, sepsis, and multiple organ failure, especially failure of the lungs, kidneys, and liver. Because of this clinical course, the need for an early recognition of AMI in the early stage, as well as an urgent, consistent clinical diagnosis and prompt revascularization are of the upmost importance.[3] But, the clinical diagnosis in early stage of AMI is very difficult. Many of the signs and symptoms associated with AMI are common to other intra-abdominal pathologic conditions, such as pancreatitis, acute diverticulitis, small-bowel obstruction, and acute cholecystitis. Ileus and sepsis are two complications that may also mask the initial signs and symptoms of AMI and make the decision making more complicated.[4]

The delay in diagnosis and treatment of acute mesenteric ischemia, as a result of the infrequent incidence and partly as a result of its non-specific clinical presentation, have contributed to the very high mortality rate.[4] Computed tomography (CT) imaging and CT angiography contribute to the diagnosis and differential management of AMI.[5] The therapeutic approach of MI includes both medical and surgical care. Although there have been tremendous advances in the medical and surgical aspects of patient care, the past 80 years have not seen an increase in the prognosis of acute mesenteric ischemia in line with these advances.[6] In this paper we present a patient with abdominal pain and inability to defecate who finally was diagnosed as having ileus and sepsis secondary to AMI and required resection of the intestine segmen.

CASE REPORT

A 59 year old man was referred to the emergency room (ER) with a diagnosis of paralytic ileus. In the ER he had good awareness with a temperature 38.9^{o}C, heart rate of 110 beats per minute, blood pressure of 100/70 mmHg, and respiratory rate of 22 times per minute. On arrival, fluid (crystalloids) resuscitation, 1 gram Ceftriaxone intravenous injection, and gastrointestinal decompression commenced and a brief history of four days of severe, intermittent abdominal pain, vomitting, absolute constipation, and fever was noted. Prior to this recent deterioration the patient had been well, with no weight loss or change in appetite. He had a past medical history of hypertension, stroke, and a 20-pack year smoking history. His medication history was only non-routine amlodipine. On inspection the patient appeared of a normal body habitus, with a BMI of 23 and examination revealed an obstructive ileus abdomen.

Blood tests from admission revealed elevated inflammatory markers (CRP 269,06 mg/L) and leucocytosis. Liver and renal function test, electrolyte serum, lipase level, amylase level, and blood gas analysis were all normal. Abdominal X-rays showed features of high obstructive ileus (shadow of intestinal gas mixed with fecal material with distribution to the pelvic cavity, coiled spring and herring bone appearance, a short air fluid level image with pathological step ladder features appears, but no visible free air outside the intestine contour). Urgent CT scan with contrast of his abdomen performed on admission revealed thickening accompanied by irregularities in the wall almost along the ileum which causes high-level narrowing of the lumen and dilation of the jejunum, duodenum, and gastric with multiple lymph nodes in the mesentery and paraaorta can be an infectious process, and also 4 cm long portal vein thrombus.

We diagnosed the patient with obstructive ileus and sepsis secondary to AMI. Surgery division consultation was done. After discussion, we agreed to perform a laparotomy. Intraoperatively, surgical colleagues found that part of the jejunum had necrosis. The resection of necrotic jejunum was done. Post operatively, we gave intravenous heparin as anticoagulant agent. The patient also had temporary fasting and total parenteral nutrition. Four day post-operatively, the patient began enteral nutrition with liquid diet. We changed intravenous heparin to subcutaneous fondaparinux 5 mg daily. Ten days post-operatively, the patient's condition improved and was discharged from hospital with oral rivaroxaban 15 mg twice per day.

Histology showed haemorrhagic infarction in sections of small bowel with ischaemic changes throughout the submucosa and muscle coat

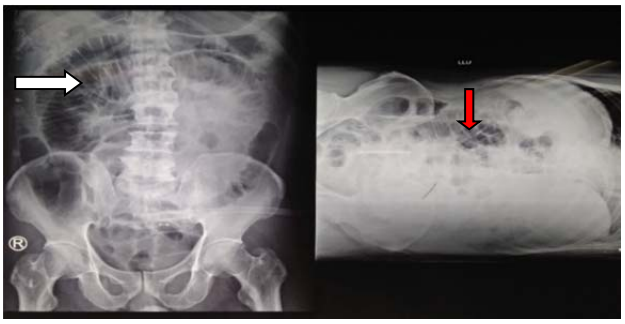

Figure 1. Abdominal X-ray photo. The white arrow show coiled spring and herring bone appearance. The red arrow show a short air fluid level image with pathological step ladder features

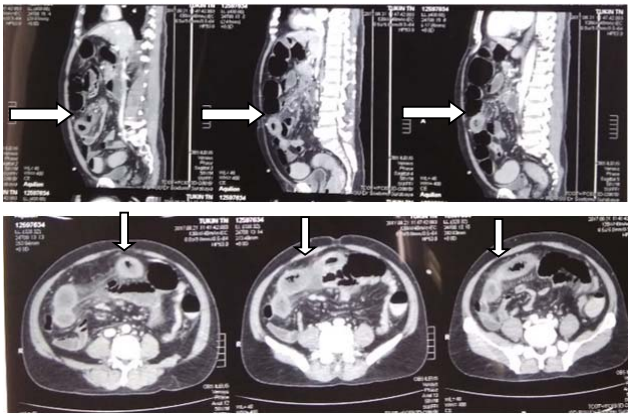

Figure 2. Abdominal Computed Tomography with contrast. The white arrows show the irregularity of intestine wall

DISCUSSION

Obstructive ileus accounts for around 15 percent of all emergency department visits for acute abdominal pain. Acute intestinal obstruction (obstructive ileus) happens when there's a disturbance within the stream of intestinal substance. This disorder can happen at any point along the gastrointestinal tract, and clinical symptoms often vary based on the degree of obstruction. Bowel obstruction is most commonly caused by intra-abdominal attachments, malignancy, or bowel herniation. The common clinical presentation incorporates nausea and vomiting, colicky stomach torment, and failure to perform flatus or bowel movements. The discoveries of classic physical examination of stomach distension, tympanic sound on percussion, and high pitched bowel sounds recommend the diagnosis. Distal obstruction permits for a bigger intestinal store, characterized by torment and distension that prevails over emesis, though patients with proximal obstruction may have negligible stomach distension but are characterized by overwhelmingly emesis. The presence of hypotension and tachycardia is a sign of a serious dehydration. Palpation of the abdomen may reveal a distended and tympanic abdomen. Be that as it may, these discoveries may not be displayed in patients with an early or proximal obstruction. Auscultation in patients with early blockage shows high pitched bowel sounds, while those with late obstruction can appear diminished bowel sounds due to the intestinal tract become hypotonic.[7]

Radiographic imaging can affirm the diagnosis, and can serve as a valuable aid on the off chance that the diagnosis is dubious. Radiographs can rapidly decide whether bowel perforation has happened. Free air can be seen over the liver in an upright or left lateral decubitus picture. In a patient with small bowel obstruction, a supine radiograph shows widened multiple loops of the small bowel, with less air within the large bowel. Patients with colon obstruction may experience large bowel dilation, with the small intestine compressed in a competent ileocecal valve condition. Perpendicular or lateral decubitus radiographs may show step ladder or air-fluid levels. These findings, along side a lack of air and stool within the distal colon and rectum, are profoundly suggestive of a mechanical bowel obstruction. In spite of the fact that radiographs are frequently the initial examination, non-contrast computed tomography is suggested if the index of suspicion is high or in case doubt continues even though radiographs are not supportive.[8] CT is sensitive for identifying high-grade obstruction (up to 90 percent in some series) and has the included advantage of deciding the cause and degree of blockage in most patients. In addition, CT can distinguish the cause of existing bowel obstruction, such as bowel volvulus or strangulation. CT discoveries in patients with bowel obstacle incorporate a loop expanding from proximal to the location of the obtruction, with the bowel distally decayed. The presence of discrete transition points helps in guiding the surgery planning. The nonappearance of contrast material within the rectum is additionally an important sign of total obtruction. For this reason, the rectal administration of contrast material should be avoided. Thickened intestinal walls and the destitute stream of contrast material into the intestinal tract demonstrate ischemia, though pneumatosis intestinalis, free intraperitoneal air, and mesenteric fat show necrosis and perforation.[9]

Management of uncomplicated obstructive ileus incorporates fluid resuscitation with correction of metabolic disorders, bowel decompression, and bowel rest. The presence of vascular complications

or perforation or failure to treat adequate bowel decompression is an indication for surgical aproach.[7]

It is additionally worth considering the other differential diagnoses of abdominal pain and diminished intestinal peristalsis. It is critical to recognize true mechanical obtruction and other causes of obstruction. One of them is acute mesenteric ischemia.[7] Acute mesenteric ischemia (AMI) is an abdominal emergency characterized by sudden cessation of intestinal bloodstream resulting in intestinal infarction. Acute mesenteric ischemia isn't an isolated clinical entity but rather involves a complex group of disorders that incorporates mesenteric artery thrombosis or embolism, mesenteric venous thrombosis, and nonocclusive mesenteric ischemia. Since of the pathophysiological heterogeneity and class differences and degree of ischemic damage, clinical and radiological appearances are diverse and often nonspecific. The key to efficient management of this syndrome follows three principles: (1) High clinical suspicion; (2) Choice of the suitable imaging strategy for building up the diagnosis; (3) Knowledge of the variables that increase the efficacy of surgery when demonstrated. This approach must be taken to induce better outcomes in managing this disease.[10] The risk factors most regularly related, in numerous case series, with this disease, were atherosclerosis (90%), heart disease (85%), systemic hypertension (85%), atrial fibrillation (75%), smoking (50%), digitalis utilize (50%) and obesity (40%).[11]

Acute mesenteric ischemia includes a complex group of disorders that incorporates embolic arterial mesenteric thrombosis, mesenteric vein thrombosis and non-occlusive mesenteric ischemia. The clinical highlights are non-specific.[10] In the early stages, hyperperistalsis, characterized by fast intestinal transit, with severe abdominal pain syndrome and no clinical relationship with other abdominal diseases, is diffuse, and its area can be related with the ischemic area; for example, in case found within the front intestine: periumbilical; center digestive tract: infraumbilical; back digestive system: pelvis. The pain does not increase with palpation and isn't related to stomach firmness. This is accompanied by nausea, vomiting (75%), and abdominal distension (25%). All of this makes early diagnosis troublesome due to likenesses with other intra-abdominal processes.[12]

The prothrombotic state, local vessel wall injuries, and venous stasis contribute to mesenteric venous thrombosis. Thrombosis can start within the *vena rectae* or the major veins. Thrombosis of the major veins is usually associated with portal vein thrombosis, though thrombosis of *vena rectae* shows as separated mesenteric vein thrombosis. The superior mesenteric vein is more frequently involved, with inferior mesenteric vein thrombosis, for unclear reason, representing only 0% to 11% of cases of mesenteric vein thrombosis.[13]

When the underlying etiology cannot be identified, the terms primary or idiopathic mesenteric venous thrombosis (MVT) is utilized. The extent of patients with primary MVT varies from 0% to 49%, with diminish in recurrence with the more broad investigation.[14] In numerous patients, more than one factor can cause thrombosis.[15] The prothrombotic state is the most common cause for patients with sisolated MVT. In contrast, localized injuries may be more frequently related with a combination of mesenteric and portal vein thrombosis.[16] Neoplasms and myeloproliferative malignancies are the most frequently diagnosed thrombophilic conditions. Stomach surgery, particularly splenectomy, can be a cause of MVT.[17] The prevalence of mesenteric and portal vein thrombosis is higher in cirrhotic patients, likely due to venous stream turbulence and the tendency to extend thrombosis.[18]

Mesenteric venous thrombosis meddling with the venous return from the digestive tract, resulting in venous swelling and ischemia. With fast and total mesenteric occlusion, there's deficient time for the development of the collateral circulation, and transmural bowel infarction may happen. The alter from typical to ischemic bowel is progressive, unlike arterial ischemia where these changes are sudden. Arterial spasm secondary to venous engorgement may occur, causing irreversible bowel ischemia, indeed when venous thrombus has been treated. Isolated mesenteric vein thrombosis results in prior and more frequent infarction progression than mesenteric vein thrombosis combined with portal vein thrombosis. Transmural infarction causes loss of intestinal mucosal integrity, permits bacterial translocation, and the potential for deadly complications such as lactic acidosis, sepsis, multi-organ failure, and death.[19]

CT scan with intravenous contrast, called CT angiography (CTA), suggests the diagnosis of primary acute mesenteric ischemia with a sensitivity of 83.3% and a specificity of 95.5%. It is considered the strategy of choice for accomplishing this diagnosis.[20] CTA could be a non-invasive examination with a positive predictive value of 100% and a negative predictive value of 94%. In case the facilities and infrastructure are lacking for CTA, a regular abdominal CT scan with contrast may be used.[21]

Table 1. Clinical features and CT findings in mesenteric ischemia[10]

Features	Arterial occlusion	Venous occlusion	Non occlusive
Incidence	60-70% of AMI	5-10% of AMI	20% AMI
Onset	Acute	Subacute	Acute or subacute
Risk factors	Arrythmia, myocardial infarction, valvular disase, atherosclerosis, prolonged hypertension	Portal hypertension, venous hypercoagulopathy, right heart failure	Hipovolemia, low heart output, digoxin, hypotension, alpha adrenergic agonists
Abdominal wall	Thin, unchanged or thickened with reperfusion	Thickened	Unchanged or thickened with perfusion
Attenuation of the abdominal wall in simple phase	Not characteristic	Low with edema; high with bleeding	Not characteristic
Enhancement of the abdominal wall in contrast phase	Reduced, absent, in target or high with reperfusion	Reduced, absent, in target or increased	Reduced, absent, with heterogenous distribution
Intestinal dilatation	Not evident	Moderate to prominent	Not evident
Mesenteric vessels	Defect or defects in arteries, arterial occlusion, SMA diameter > SMV	Defect or defects in veins, congestive veins	No defects, arterial constriction
Mesentery	Homogenous until an infarction occurs	Heterogenous with ascites	Homogenous until an infarction occurs

SMA: superior mesenteric artery; AMI: acute mesenteric ischemia; SMV: superior mesenteric vein

Medical management of acute mesenteric ischemia incorporates enhancement of the general condition and anticoagulation. Pain control, bowel rest, and fluid and electrolyte substitution ought to be started for the acute presentation. Red cell transfusions may be required for gastrointestinal bleeding and nasogastric insertion for patients with severe abdominal distension, ileus, and nausea or vomiting. Broad-spectrum antibiotics are applicated for patients with sepsis due to bacterial translocation from intestinal infarction.[22]

Nonsurgical management of patients with acute mesenteric ischemia ought to be considered as the first treatment step and correction of the risk factors most frequently associated in the different case series with this disease: atherosclerosis, heart disease, systemic hypertension, atrial fibrillation, smoking, digitalis use, and obesity.[23] If there are intense complications such as perforation, peritonitis, and intestinal necrosis, a surgical approach is indicated. Cerebral, hepatic, or renal infarction may be a finding that proposing a poor prognosis in patients with intestinal ischemia even without intestinal vascular involvement. These varieties depend on the pathogenesis of ischemia, duration, area, and expansion of the intestine, collateral circulation, additional infection, or whether there is a perforation or not.[24]

In this patient, portal vein thrombus was also found. The objectives of treatment for acute portal vein thrombosis without cirrhosis are to set up complete patency of the portal vein, thereby preventing the development of chronic portal vein thrombosis, and to decrease the risk of constituting thrombus into the mesenteric vein and prevent mesenteric ischemia and infarction. Randomized controlled studies about the adequacy of most forms of treatment for portal vein thrombosis are lacking. Utilize of unfragmented heparin or, ideally, low-molecular-weight heparins such as enoxaparin or dalteparin, with a consequent transition to oral warfarin, is the foremost common approach to anticoagulants.[25]

The ideal duration of anticoagulation is controversial and can run from 6 months to a lifetime anticoagulant, depending on the etiology of vascular thrombosis. For most patients with systemic etiology, life-long anticoagulants are recommended in case there are no contraindications. Portal vein recanalization can be affirmed by Doppler ultrasound examination after 6 months of anticoagulation. Combined information from retrospective studies recommends that rates of recanalization after 6 months of anticoagulation can vary from total recanalization in 50% or partial recanalization in 40% to the failure of recanalization in up to 10% of patients. In a single prospective study of 105 patients with acute portal vein thrombosis, 44% of patients accomplished portal venous flow patency, and mortality was 2% at 1-year follow-up. For patients with partial or incomplete recanalization after 6 months of treatment, it is unclear whether proceeded anticoagulants give an extra advantage. Spontaneous recanalization of portal vein vessels has been reported in some patients.[26]

Nutritional support improves dietary status and optimizes clinical results in critically ill patients. Studies on postoperative nutritional support have appeared diminished morbidity and length of stay in hospital. Conventional treatment after bowel resection with intravenous liquids as it were until the presence of flatus, as a rule, causes starvation, primarily since of postoperative ileus concern. This can be based on the presumption that oral feeding may not be tolerated in the presence of ileus and the integrity of the recently made anastomosis may be influenced. However, small bowel motility recoups 6-8 hours after surgical trauma and moderate absorption capacity is present even

when there is no normal peristalsis. This proposes that postoperative administration of enteral nutrition in patients experiencing gastrointestinal resection is secure and well-tolerated indeed when begun inside 12 hours after surgery. The foremost common side impacts are gastrointestinal side effects, such as stomach spasms and bloating.[27]

The most important message linked to this case presentation is that acute mesenteric ischemia may mimic other diseases such as ileus. From an internal medicine point of view, it is advisable to conduct a quantitative analysis giving a specific value to the different findings, including risk factors, physical examination, laboratory studies, and image findings, to determine the risk of acute mesenteric ischemia in a patient with acute abdominal pain syndrome.

REFERENCES

1. Roussel A, Castier Y, Nuzzo A, Pellenc Q, Sibert A, Panis Y, et al. Revascularization of acute mesenteric ischemia after creation of a dedicated multidisciplinary center. J Vasc Surg 2015;62:1251-6.
2. Herbert GS, Steele SR. Acute and chronic mesenteric ischemia. Surg Clin North Am 2007;87:1115–34.
3. Luthera B, Mamopoulos A, Lehmann C, Klard E. The ongoing challenge of acute mesenteric ischemia. Visc Med 2018;34:217–23.
4. Oldenburg WA, Lau LL, Rodenberg TJ, Edmonds HJ, Burger CD. Acute mesenteric ischemia: a clinical review. Arch Intern Med 2004;164:1054–62.
5. Mastoraki A, Mastoraki S, Tziava E, Touloumi S, Krinos N, Danias N et al. Mesenteric ischemia: Pathogenesis and challenging diagnostic and therapeutic modalities. World J Gastrointest Pathophysiol 2016;7:125-30.
6. Assar AN, Zarins CK. Acute mesenteric ischaemia: facts and perspectives. British Journal of Hospital Medicine 2008;69:634-40.
7. Jackson PG, Raiji M. Evaluation and management of intestinal obstruction. Am Fam Physician 2011;83:159-65.
8. Stoker J, van Randen A, Laméris W, Boermeester MA. Imaging patients with acute abdominal pain. Radiology 2009;253:31-46.
9. Furukawa A, Yamasaki M, Furuichi K, Nagata T, Takahashi M. Helical CT in the diagnosis of small bowel obstruction. Radiographics 2001;21:341-355.
10. Motta-Ramírez GA, Sánchez-García JC, Ontiveros-Rodríguez A, López-Ramírez MA, Rebollo-Hurtado V, García-Ruiz A et al. Acute mesenteric ischemia: an emergency that requires a comprehensive diagnostic approach. Anales de Radiología México 2015;14:66-88.
11. Byard RW. Acute mesenteric ischaemia and unexpected death. J Forensic Leg Med 2012;19:185-90.
12. Florian A, Jurcut R, Lupescu I, Grasu M, Croitoru M, Ginghină C. Mesenteric ischemia – a complex disease requiring an interdisciplinary approach. A Review of the current literature. Rom J Intern Med 2010;48:207–22.
13. Thatipelli MR, McBane RD, Hodge DO, Wysokinski WE. Survival and recurrence in patients with splanchnic vein thromboses. Clin Gastroenterol Hepatol 2010;8:200-5.
14. Abu-Daff S, Abu-Daff N, Al-Shahed M. Mesenteric venous thrombosis and factors associated with mortality: a statistical analysis with five-year follow-up. J Gastrointest Surg 2009;13:1245-50.
15. Orr DW, Patel RK, Lea NC, Westbrook RH, O'Grady JG, Heatonet ND, et al. The prevalence of the activating JAK2 tyrosine kinase mutation in chronic portosplenomesenteric venous thrombosis. Aliment Pharmacol Ther 2010;31:1330-6.
16. Kumar S, Kamath PS. Acute superior mesenteric venous thrombosis: one disease or two? Am J Gastroenterol 2003;98:1299-1304.
17. Cenedese A, Monneuse O, Gruner L, Tissot E, Mennesson N, Barth X. Initial management of extensive mesenteric venous thrombosis: retrospective study of nine cases. World J Surg 2009;33:2203-8.
18. Tripodi A, Mannucci PM. The coagulopathy of chronic liver disease. N Engl J Med 2011;365:147-56.
19. Singal AK, Kamath PS, Tefferi A. Mesenteric venous thrombosis. Mayo Clin Proc 2013;88:285-94.
20. van den Heijkant TC, Aerts BAC, Teijink JA, Buurman WA, Luyer MDP. Challenges in diagnosing mesenteric ischemia. World J Gastroenterol 2013;19:1338-41.
21. Horton KM, Fishman EK. Multi-detector row ct of mesenteric ischemia: can it be done? Radiographics 2001;21:1463–73.
22. Grisham A, Lohr J, Guenther JM, Engel AM. Deciphering mesenteric venous thrombosis: imaging and treatment. Vasc Endovasc Surg 2005;39:473-9.
23. Ravipati M, Katragadda S, Go B, Zarling EJ. Acute mesenteric ischemia: a diagnostic challenging clinical practice. Practical Gastroenterology 2011;38:35-43.
24. Singer A, Handler BJ, Simmons MZ, Baker SR. Acute small bowel ischemia: Spectrum of computed tomographic findings. Emerg Radiol 2000;7:302-7.
25. de Franchis R. Evolving consensus in portal hypertension. Report of the Baveno IV consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol 2005; 43:167-176.
26. Plessier A, Darwish-Murad S, Hernandez-Guerra M, Consigny Y, Fabris F, Trebicka J, et al. A prospective multicentric follow-up study on 105 patients with acute portal vein thrombosis (PVT): results from the European Network for Vascular Disorders of the LIver (EN-Vie). Hepatology 2007;46:310A.
27. Braga M, Gianotti L, Gentilini S, Liotta S, Di Carlo V. Feeding the gut early after digestive surgery: results of a nine-year experience. Clin Nutr 2002;21:59-65.